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PERENNIAL MYCELIUM IN SPECIES OF PERONOSPORACEAE RELATED TO PHYTOPHTHORA INFESTANS

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INTRODUCTION

Phytophthora infestans having been found to be perennial in the Irish potato (*Solanum tuberosum*), the question naturally arose as to whether other species of Peronosporaceae survive the winter in the northern part of the United States in the mycelial stage. As shown in another paper (13),[1] the mycelium in the mother tuber grows up the stem to the surface of the soil and causes an infection of the foliage which may result in an epidemic of late-blight.

Very little is known about the perennial nature of the mycelium of Peronosporaceae. Only two species have been reported in America: *Plasmopara pygmaea* on *Hepatica acutiloba* by Stewart (15) and *Phytophthora cactorum* on *Panax quinquefolium* by Rosenbaum (14). Six have been shown to be perennial in Europe: *Peronospora schachtii* on *Beta vulgaris* and *Peronospora dipsaci* on *Dipsacus fullonum* by Kühn (7, 8); *Peronospora alsinearum* on *Stellaria media*, *Peronospora grisea* on *Veronica hederaefolia*, *Peronospora effusa* on *Spinacia oleracea*, and *Atriplex hortensis* by Magnus (9); and *Peronospora viticola* on *Vitis vinifera* by Istvanffi (5).

Many of the hosts of this family are annuals, but some are biennials, or, like the Irish potato, are perennials. Where the host lives over the winter, it is interesting to know whether the mycelium of the fungus may also live over, especially where the infection has become systemic and the mycelium is present in the crown of the host plant. The absence or sparse production of oospores in some of the species of Peronosporaceae, coupled with the appearance of the fungus as soon as the host puts out foliage in the spring, suggests that the mycelium may play an important

[1] Reference is made by number to "Literature cited," p. 68–69.

rôle in bridging over the winter. This paper gives the results of experiments and observations which show that in the Northern States species of the Peronosporaceae which have perennial mycelium are common and that the mycelium may live from one growing season to another in the living diseased host tissues.

In several of these experiments the locality where infected plants were growing was marked in the autumn and the plants collected from time to time during the winter and early spring, after which they were allowed to revive in the greenhouse and a careful watch kept for any evidence of fruit of the fungus. In other cases the underground parts of infected plants were taken in the spring and planted in steam-sterilized soil in the greenhouse, and when the shoots came through the ground conditions were made favorable for the sporulation of the fungus. In still other cases the presence of the mycelium in perennial parts of the host was determined microscopically.

PERONOSPORA PARASITICA

Late in the fall of 1910 and 1911 it was observed that young plants of *Lepidium virginicum* in the vicinity of Madison, Wis., were very generally infected with *Peronospora parasitica* and that the tissues of these plants contained few or no oospores, although they were produced in abundance in the summer when the host tissues were dying. Plants of *Lepidium* sp. always form a rosette of leaves in the late fall, and some of these remain alive through the winter.

In the fall of 1911 two patches of Lepidium plants, about 50 per cent of which were infected with *Peronospora parasitica*, were marked so that they might be easily found during the winter. One was on the side of a short incline made by dumping several loads of soil in a heap and the other on the parking of a city drive in Madison. Both patches were well exposed during the winter of 1911–12, which was unusually severe, there being no covering of snow on the former at any time and the latter being covered only a part of the time.

After the first killing frost, which, according to the Weather Bureau, occurred on October 24, infected plants of *Lepidium virginicum* were collected at various times during the winter. Beginning on October 30, a test was made of the germination of the conidia of *Peronospora parasitica* growing on *Lepidium virginicum*. Although when alive the conidia of this fungus usually germinate profusely within 2 to 3 hours and always within 24 hours, no germination occurred in this test, although exposed to favorable conditions for 48 hours. This coincides with what is known of the behavior of the spores of other species—e. g., *Cystopus candidus* (Melhus, 10)—and excludes the possibility of these conidia becoming a source of further infection. A careful search for oospores was made after October 30 in a large number of infected plantlets, but none was found.

The first collection of plants of *Lepidium virginicum*, numbering about 20, was made on November 5, enough soil being taken up with each plant to keep the roots from being disturbed. The plants were taken to the greenhouse and transplanted in two flats, or shallow boxes, and on November 6 each box was covered with a low bell jar to keep the air moist, a condition favorable for the sporulation of the fungus. An examination of the plants next day showed but 2 inactive, the leaves of the other 18 being turgid and expanded in the normal way. It also showed that 2 of the plants were covered with a white glistening growth, which on microscopic examination was found to be the spores and conidiophores of *Peronospora parasitica*. The following day this fungus was found sporulating on 3 additional plants, and 8 days after the plants had been collected it was found fruiting on some portion of 12 of the 18 living. Although kept under observation for 6 weeks, the remaining six plants were free from infection, which showed that it did not take place under the conditions in which they were held in the greenhouse.

On December 14 another collection of plants of *Lepidium virginicum* was taken from the patch on the parking near the drive, the soil at that time being frozen 6 inches deep. A block of soil on which there were 18 of the plants was chopped loose and placed in a flat in the greenhouse, and after being allowed to thaw out for 24 hours was covered with a glass house. On December 17, 3 days after the plants were brought into the greenhouse, 1 was nearly covered with conidiophores and spores of *Peronospora parasitica*, the next day 4 more showed fruit of the fungus, and at the end of the sixth day an additional plant, or 6 in all, showed fruiting of the fungus, indicating that at least that number was infected when collected (Pl. III, fig. 2, *A*). The fungus fruited on both sides of the leaves and also on the new leaves developing from the crown, though not as abundantly on these as on the older leaves.

Besides the collections of November 5 and December 14, 4 others, or a total of 102 plants, were brought into the greenhouse from the 2 patches during the dormant period of the host plant. In the case of several of these collections *Peronospora parasitica* sporulated on some of the plants 2 days after their transfer to the greenhouse, but usually the disease did not appear before 3 to 5 days and, when the infection was weak, not before 8 days after the transfer. Table I gives date of collection, number of plants in each collection, date of first evidence of Peronospora, number of days required for the fungus to sporulate, and number of plants on which the disease appeared.

TABLE I.—*Record of six collections of plants of Lepidium virginicum infected with Peronospora parasitica*

Date of collection.	Number of plants.	Date of sporulation.	Number of days required for sporulation.	Number of plants on which fungus sporulated.
1911.				
Nov. 5	20	Nov. 8	3	12
Dec. 14	18	Dec. 17	3	6
Dec. 18	12	Dec. 20	2	6
1912.				
Feb. 22	11	Feb. 27	5	1
Mar. 6	24	Mar. 10	4	7
Mar. 25	17	Mar. 27	2	9

As shown by Table I, 41 plants, or about 40 per cent of the collections, were infected before their transfer to the greenhouse.

It might be supposed that oospores produced the previous year were in the soil immediately around and adhering to the plants collected and that when warmed up in the greenhouse these germinated and produced the infections noted. To test this possibility, 25 leaves were collected from the plants in the two patches, washed very thoroughly in running water, and placed in a moist chamber, while 25 other leaves were collected from the same plants, and without being washed were placed under similar conditions as controls. In both cases the fungus sporulated after three days, and, although much less than when the leaves were on the plant, the sporulation produced sufficient conidiophores to be plainly visible to the naked eye, a growth which could probably not be produced by oospores.

Besides this evidence that *Peronospora parasitica* renews itself by means of mycelium as well as oospores, the writer failed to germinate oospores after repeated attempts. He has also shown (11) that *Peronospora parasitica* on *Lepidium virginicum* can be collected at any time during the winter and early spring, brought into the greenhouse, and made to fruit. Moreover, there can be no doubt that the sporulation on the plant collections at Madison was due to living mycelium in the host tissue.

CYSTOPUS CANDIDUS

Lepidium virginicum is attacked not only by *Peronospora parasitica* but also by *Cystopus candidus*, a fungus which can undoubtedly propagate itself from year to year by mycelium remaining dormant in the living host tissues through the winter. As is well known, these two fungi often infect a plant simultaneously, as was the case of some of the plants from the parking near the drive. In the collections made on December 14, 1911, one plant showed white pustules of *Cystopus candidus* on December 17, three days after the plants were collected. The following day

two additional plants showed white pustules of this fungus and also spores of *Peronospora parasitica*, the number of pustules increasing on the lower side of the leaves until many were well spotted. Two plants in the collection made on February 22 bore white pustules within three days after they were taken into the greenhouse, showing that they were infected with *Cystopus candidus* and that the fungus was alive in the tissues in late winter (Pl. III, fig. 1). Again, in the collection made on March 25 one plant developed pustules of *Cystopus candidus* and conidiophores and spores of *Peronospora parasitica* four days after being transferred to the greenhouse.

Cystopus candidus is also a very common parasite on *Capsella bursa pastoris*, a plant that may become a winter annual. In the fall of 1911 a patch of plants of *Capsella bursa pastoris*, many of which were infected with *Cystopus candidus*, was marked; and on March 30, 1912, 25 plants were collected and treated in the same way as the plants of *Lepidium virginicum* infected with *Peronospora parasitica*. After two days the plants began to show signs of life; and at this time they were covered with a small glass house. Three days later white pustules were discovered on one leaf; and the following day, or six days after the plants were brought in, white pustules developed on other leaves of the same plant.

On April 5, 1912, just as the ground thawed out, another collection, consisting of 76 plants, was made. Four days after, or on April 9, there were white pustules on four of the plants. Except in the case of one large leaf, which was probably produced early the preceding fall, the pustules were all on the youngest leaves, which indicates that the mycelium can winter over in leaves of plants of *Capsella bursa pastoris* that live through the winter. The fact that the youngest leaves were infected suggested crown infection; and later this proved to be the case, all of the leaves growing from certain plants being infected as soon as they appeared, while the leaves growing from certain others remained free from infection. On April 10 white pustules appeared on two other plants, making a total of six infected plants in the second collection. As soon as the plants of *Capsella bursa pastoris* in the marked patch started to grow in the spring some of them showed infection with *Cystopus candidus*, which developed like the infections studied in the greenhouse. From these experiments it will be seen that the mycelium of *Cystopus candidus* in the tissues of the host remains alive through the winter.

In the fall of the year *Cystopus candidus* becomes systemic in the tissues of *Sisymbrium officinale* and *Brassica nigra* also. So far these two host plants have not been followed through from fall to spring, but, like the plants of *Lepidium virginicum* and *Capsula bursa pastoris*, both may become winter annuals, as is well known.

PERONOSPORA FICARIAE

On May 10, 1911, *Peronospora ficariae* was very prevalent on *Ranunculus fasciculatis* in the vicinity of Madison. This fact, coupled with De Bary's (3) statement in connection with his discussion of the perennial nature of mycelium of *Phytophthora infestans*, that *Peronospora ficariae* is perennial in the tissues of *Ranunculus ficaria*, led the writer to determine whether it survives the winter in the mycelial stage on *Ranunculus fasciculatis* also. Eighteen plants, very generally infected with the disease, were staked on the date above mentioned so that they could be readily located throughout the winter and following spring. On February 2, 1912, five of the plants were chopped out of the frozen ground and carried into the greenhouse, where the adhering soil was allowed to thaw out and was removed from the fascicled roots, after which the roots were carefully washed until free from soil and then transplanted in greenhouse soil. The plants, two of which refused to grow, started very slowly, the first one coming up on March 3, and two others the following day. The young plants were chlorotic, distorted, and yellowish green, but there was no evidence of *Peronospora ficariae* present until they had been held under small bell jars for 24 hours, after which the fungus present on the deformed leaves fruited profusely, showing plainly that the fungus was alive in the host tissues during the winter.

The 13 plants that were left in the marked space from which the 5 were taken were also watched carefully after they began to come up. On April 5 five appeared, and these were covered with small bell jars. On the following day conidiophores and spores of *Peronospora ficariae* were collected from the underside of the leaves, showing that in this case also the plants were infected before they reached the surface of the soil. The results of these experiments confirm De Bary's (3) statement and also show that *Peronospora ficariae* is perennial not only in *Ranunculus ficaria* but also in *Ranunculus fasciculatis*.

PERONOSPORA VICIAE

Peronospora viciae occurs on several of the legumes. On May 11, 1913, the writer found it to be quite abundant on *Vicia sepium*, a perennial common in the District of Columbia. At that time about 25 per cent of the plants, which were from 4 to 6 inches high, were infected with the disease, the fungus sporulating profusely and the plants giving every evidence of systemic infection. The location of these plants was staked off on the date above mentioned and the patch kept under observation. On April 5 the following spring the plants started to come up, the tallest being only 2 inches, and at this early stage nine were found to be systemically infected. It was not uncommon to find a healthy and a diseased plant within 2 inches of each other. If infection was caused by oospores or conidia, it is difficult to understand why the infection was not general

in the patch and why plants growing near each other should be infected in some cases and not in others.

As the host is a perennial, as infection by *Peronospora viciae* is systemic, and as oospores are produced only sparingly, if at all, on *Vicia sepium*,[1] it seems very probable that the mycelium survives the winter in the living tissues of the host.

PLASMOPARA HALSTEDII

In the spring of 1911 *Plasmopara halstedii* was found to be very abundant on some young plants of *Helianthus diversicatus* about 6 inches high. The plants were somewhat dwarfed, chlorotic, and well covered with conidiophores, giving every evidence of systemic infection. The location of the infected plants was marked and observations made during the winter and spring of 1912.

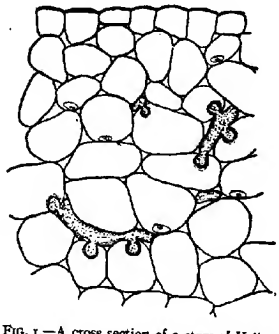

FIG. 1.—A cross section of a stem of *Helianthus diversicatus* which is infected with *Plasmopara halstedii*. The mycelium is shown in the cortex at the junction of the stem with the rhizome of the host.

Fourteen of the plants that were very generally infected were staked, and on January 4, three of these were chopped out of the ground and transplanted in the greenhouse in exactly the same way as were the Lepidium plants infected with *Peronospora parasitica*. Each of these rhizomes produced a chlorotic shoot which was covered with spores of *Plasmopara halstedii*. On March 4 four more were brought into the greenhouse. One of these rotted in the soil, but each of the others produced a shoot, which showed infection as soon as it appeared above ground. The remaining seven of the fourteen staked were left in the patch and kept under observation. On May 10, when they were 3 to 6 inches high, all were found to be infected with *Plasmopara halstedii*, except one plant, which was entirely free from infection, as were many others in the immediate vicinity. Two of these plants were now dug up, and portions of the stems at their junction with the rhizomes were fixed in various strengths of Flemming's killing fluid. Paraffin sections cut from this material and stained showed abundant mycelium in all parts of the stem except the fibrovascular bundles, the mycelium being entirely intercellular with globular haustoria extending into the cells, as shown in figure 1. The presence of the mycelium in the stem at its junction with the rhizome shows that the infection was systemic and probably came from the rhizome in the beginning.

[1] The writer searched many times in the tissues of all stages of maturity for resting spores, but without success.

The remaining five of these seven infected plants were carefully dug up, the stems cut off at their junction with the rhizomes, washed very clean with a brush, and disinfected in corrosive sublimate for five minutes. After this they were planted in steam-sterilized soil in the greenhouse, in which there had never been any *Plasmopara halstedii*. On May 23 two shoots broke through the ground; and three days later, when one was 1 inch and the other 2 inches high, they were covered with jelly glasses in order to keep the atmosphere moist. On this date the initial leaves appeared chlorotic, but no spores of *Plasmopara halstedii* could be found. The next day the lower surfaces of the leaves were almost covered with a glistening white coat of conidiophores and spores, which on microscopic examination were found to be the conidia of *Plasmopara halstedii*. Of the three remaining rhizomes, two failed to come up, while the third sent up a spindly shoot on June 5. This shoot was treated in the manner already described and the fungus fruited in the same way.

This experiment showed that the diseased plants grown in the greenhouse manifested the same symptoms as those grown in the open. It also showed that the mycelium of *Plasmopara halstedii* may be present in the rhizome of *Helianthus diversicatus*, and this, coupled with the observations described, strongly suggests that *Plasmopara halstedii* is perennial in the rhizomes of *Helianthus diversicatus*.

CONCLUSIONS

As seen from these investigations, several species of the Peronosporaceae live over from one growing season to another by at least two means: Resting spores and perennial mycelium. As is well known from the excellent studies of De Bary (2), the oospores germinate after a rest period either by zoospores or germ tubes and cause the infection of plant tissues. Because of their extremely ephemeral nature, the conidia hardly merit consideration as resting organs, but, nevertheless, they may under certain conditions function as such. If a fungus has two or more annual host plants, it may spread to one or more by conidia after primary infection has resulted from oospores on one; or the fungus may be perennial in one host and spread to another by conidia borne on the former—e. g., *Phytophthora infestans* on the potato and tomato.

The species of Peronosporaceae known to have perennial mycelium are given in Table II.

TABLE II.—*Species of Peronosporaceae having perennial mycelium*

Name of fungus.	Name of host.	Authority.
Phytophthora infestans	*Solanum tuberosum*...	De Bary (1), 1861, Bonn, Germany.
Do	do	Jensen (6), 1887, Nerilly, France.
Do	do	Melhus (12), 1913, Houlton, Me.
Phytophthora cactorum	*Panax quinquefolium*	Rosenbaum (14), 1914, Ithaca, N. Y.
Cystopus candidus	*Capsella bursa pastoris*	Melhus (12), 1913, Madison, Wis.
Do	*Lepidium virginicum*	Do.
Plasmopara viticola	*Vitis vinifera*	Istvanffi (5), 1904, Budapest, Austria.
Plasmopara pygmaea	*Hepatica acutiloba*	Stewart (15), 1910, Ithaca, N. Y.
Plasmopara halstedii	*Helianthus diversicatus*	Melhus (12), 1913, Madison, Wis.
Peronospora dipsaci	*Dipsacus fullonum*	Kühn (8), 1875, Halle, Germany.
Peronospora schachtii	*Beta vulgaris*	Kühn (7), 1872, Halle, Germany.
Peronospora alsinearum	*Stellaria media*	Magnus (9), 1888, Berlin, Germany.
Peronospora grisea	*Veronica hederaefolia*	Do.
Peronospora effusa	*Spinacia oleracea*	Do.
Do	*Atriplex hortensis*	Do.
Peronospora ficariae	*Ranunculus ficaria*	De Bary (3), 1876, Bonn, Germany.
Do	*Ranunculus fascicularis*	Melhus (12), 1913, Madison, Wis.
Peronospora parasitica	*Lepidium virginicum*	Do.
Peronospora viciae	*Vicia sepium*	Melhus (13), 1915, District of Columbia.
Peronospora rumicis	*Rumax acetosa*	De Bary (3), 1876, Bonn, Germany.

There can be no doubt that the mycelium of several species of Peronosporaceae may become perennial. Of course this can take place only when the host is a winter annual, biennial, or perennial, and quite generally infected. Such plants may live through the winter and renew activity in the spring, when the fungus may sporulate and spread the disease.

The perennial nature of the mycelium of other species of the genus Phytophthora has not been studied critically, but there is reason to believe that *Phytophthora infestans* is not the only one that may become perennial. In many cases other species produce oospores prolifically. Butler and Kulkarni (4) believe that on Colocasiae *Phytophthora colocasiae* may survive the dry seasons of India in the mycelial stage. Another case of perennial mycelium is that of *Phytophthora cactorum* on ginseng (*Panax quinquefolium*), a perennial having a fleshy root, described by Rosenbaum (14). The Phytophthora fungus flourishes on the roots, and, according to this author (14), can spread from the roots up the stem to the surface of the soil, and produce conidia which infect the foliage, a case very analogous to *Phytophthora infestans*.

Table II shows that, so far as known, only one species of Cystopus has perennial mycelium—that is, *Cystopus candidus* on two hosts, *Lepidium virginicum* and *Capsella bursa pastoris*. Both of these plants may be either annuals or winter annuals, and in both the fungus may become systemic and may survive the winter, provided the host plants live. Unlike *Phytophthora infestans*, *Cystopus candidus* produces oospores pro-

fusely in these two host plants after they mature or are killed by the parasite, but the writer has been unable to find oospores in the young plants during the fall, and this agrees with Magnus's (9) report that oospores are not produced in the seedling plants of spinach infected with *Peronospora effusa* in the fall. Magnus also states that the same is true in the case of *Stellaria media* and *Veronica hederaefolia* infected with *Peronospora alsinearum* and *Peronospora grisea*, respectively.

The number of species of the genus Peronospora that may survive the winter in the mycelial stage are more numerous. Table II shows nine. Careful study is in progress in regard to the remaining species of this genus. As also shown in this table, there are three species of Plasmopara which may survive the winter in this stage, and this number, the writer is confident, will be increased by further studies.

SUMMARY

(1) There are at least several species of Peronosporaceae belonging to four genera that may be perennial in the tissues of their hosts, the mycelium passing the winter either in the aerial or the underground organs of winter annuals, biennials, or perennials.

(2) *Phytophthora infestans* is not an exception in the family to which it belongs as regards perennial mycelium.

(3) The rôle of the mycelium of *Phytophthora infestans* in the tubers of its host is not an unusual one. It may grow from the tubers up the stem to the surface of the soil, sporulate, cause foliage infection, and bring about an epidemic of the disease.

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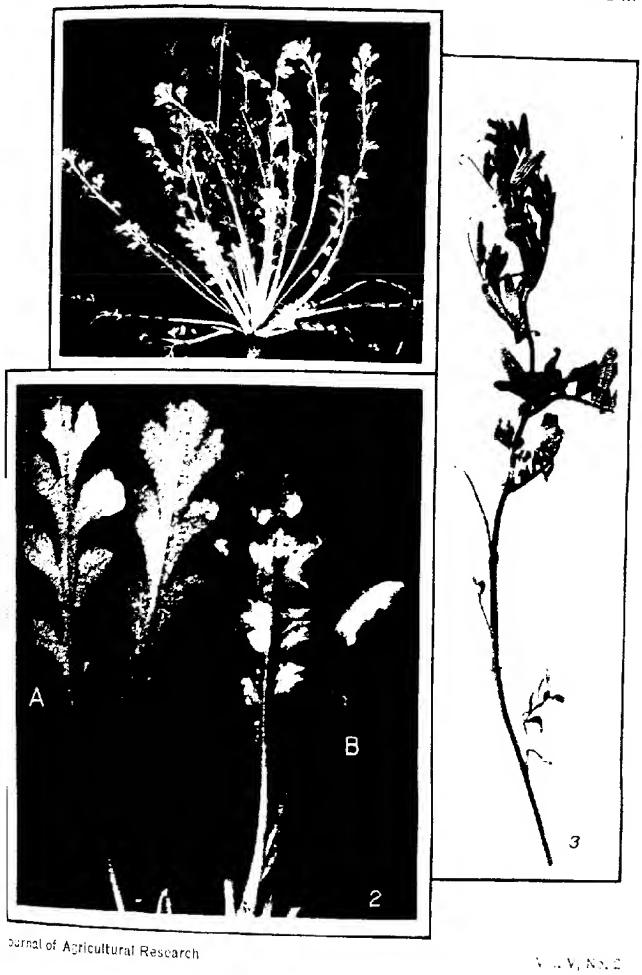
PLATE III

Fig. 1.—*Cystopus candidus* on *Lepidium virginicum*. This plant was chopped out of the frozen ground on February 22, 1911, and brought into the greenhouse. Three days later white pustules of *Cystopus candidus* began to appear on the leaves.

Fig. 2.—*A*, The two leaves at the left show the amount of sporulation of *Peronospora parasitica* on leaves of *Lepidium virginicum; B*, the two leaves at the right show *Cystopus candidus* fruiting on leaves of *Capsella bursa pastoris*. The pustules developed from mycelium alive in the plants in the winter of 1911.

Fig. 3.—*Peronospora viciae* on *Vicia sepium*. A systematic infection of the downy mildew collected on April 15, 1914, in the District of Columbia. This plant was badly infected when coming through the ground.


A
B
2
3

HIBERNATION OF PHYTOPHTHORA INFESTANS IN THE IRISH POTATO

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INTRODUCTION

How *Phytophthora infestans* perpetuates itself from year to year has been studied ever since Unger in 1847 (34) [1] finally proved that the fungus causing the disease is a species of Peronospora. No sooner had this fact been established than students began searching for resting organs like those so common in other species of Peronosporaceae. As is well known, progress was slow, and the question as to whether *P. infestans* does or does not have oospores ended in a controversy between W. G. Smith (30) and De Bary (4) in the early seventies of the last century. The outcome is too well known to need repetition; suffice it to say that De Bary's negative evidence has been generally accepted.

Recently the oospore question has been taken up anew and bodies resembling oospores have been found by Jones (15, 16, 17), Clinton (9), and Pethybridge and Murphy (27) in pure cultures of the fungus. Although no direct claims that similar bodies exist in the potato plant (*Solanum tuberosum*) have been made, these recent investigations have at least weakened the perennial-mycelium theory, which probably was first advanced by Berkeley in 1846 (5). Like many of the botanists during the first half of the last century, Berkeley unfortunately submitted no experimental evidence to support his contention. The credit of first submitting such evidence belongs to De Bary, who in 1861 in an interesting paper (1) showed that the conidia can not live over winter; that no relation exists between the mycelium of *P. infestans* and of the saprophytes that occur on diseased tubers; that it is impossible to infect potatoes with any of the Peronosporaceae that occur on plants common about potato fields; and that the potato fungus is able to spread from diseased seed tubers up into the shoots, sporulate, and renew infection on the foliage.

About 10 years later, Scholtz, Bretschneider, Peters, and Reess took up for the "Central Commission für das Agrikulturchemische Versuchswesen" the problem how *P. infestans* perpetuates itself. They were unable to confirm De Bary (1), and Pringsheim (29), who sum-

[1] Reference is made by number to "Literature cited," p. 100–102.

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marized their work, ~~[illegible]~~ the alternate-host theory as a final resting place for this unsolved problem.

The fact that none of these investigators was able to confirm De Bary (1) and the announcement of W. G. Smith in 1875 (30) that he had found the oospores of *P. infestans* doubtless influenced the Royal Agricultural Society to ask De Bary to again take up a study of how this fungus perpetuates itself. In a report to the Society in 1876 De Bary (4) makes the following general statement (p. 265), based on his observations and experiments, which shows plainly his thorough understanding of the habits of *P. infestans*.

> I was, perhaps, the first to call attention distinctly to the fact that the mycelium of *Phytophthora*, like that of parasites living in many other perennial plants, can be perennial in the surviving parts of the hosts, i. e. in the case of the potato, in its tubers.

It has already been pointed out that Berkeley (5) first suggested that the mycelium of *P. infestans* is perennial in the potato tuber. Many attempts have since been made by Jensen (14), Boehm (6), Smorawski (32), Hecke (12), and others to duplicate De Bary's (4) experiments both in the laboratory and in the field, but no one except Jensen has obtained confirmatory evidence, and his evidence has failed to strengthen the perennial-mycelium theory.

Naturally the accumulated negative evidence has led many to doubt the perennial capacity of the mycelium and to substitute widely different hypotheses. At least six theories as to the yearly advent of this disease have been advanced at various times: (1) That the mycelium lives over winter in the soil; (2) that mycelium is perennial in the diseased tuber; (3) that resting spores are produced which function in renewing infection; (4) that the mycelium is latent in the potato plant; (5) that the fungus fruits on the parent tuber in the soil and the spores reach the surface and cause infection of the foliage; and (6) that sclerotia-like bodies or a mucoplasm gives rise to infection. The second of these is the only one supported by any amount of experimental data, the other five being based chiefly on negative evidence, of which there is considerable.

In this paper are recorded data obtained in the laboratory and field supporting the perennial-mycelium theory.

EXPERIMENTAL STUDIES

The present study has to do largely with the function of the mycelium of *P. infestans* in infected tubers and its relation to the progeny of the host plant. The spread of the mycelium in tubers and sprouts was considered first and followed by further experiments to determine the relation of the mycelium to the shoots and young plants. Later, infected tubers were planted in the field and the progeny watched for any evidence of the disease.

RELATION OF MOISTURE AND TEMPERATURE TO THE SPREAD OF THE MYCELIUM OF PHYTOPHTHORA INFESTANS IN THE TUBERS

In order to learn something as to the influence of environmental factors upon the spread of the mycelium, tubers naturally infected with *P. infestans* were taken and the boundary line of the infected area marked with india ink. Thirty-three tubers were thus treated and buried in steam-sterilized sand in boxes 6 inches deep. The sand was kept continuously well moistened. The infected areas included from 20 to 90 per cent of the total surface area of the tubers, but there were at least two living eyes. The boxes with these tubers were kept in a greenhouse where the temperature was held at 16° C. at night and 22° in the daytime. After 12 days the tubers were all taken up and the boundary line between the sound and infected areas again traced. In every case the fungus was found to have made progress at some point on the tuber, but the progress was not uniform, the lines coinciding at some points and diverging as much as 1 inch at others. The spread seemed to have been more rapid in the vicinity of the eyes, although this was not always the case. On 7 of the 33 tubers sprouts were found that were infected with *P. infestans*, which at the time of planting, 12 days earlier, were sound.

Again the tubers were planted in the moist sand and allowed to develop for 9 days more. When dug up this time, the fungus was found to have spread over all the remaining sound surface area, except in the case of two tubers, and even in these it had made material growth. This time four more sprouts were found to be infected with *P. infestans*, its presence being proved by cutting the sprouts off and holding them for 24 hours in a moist atmosphere, the fungus in the meantime fruiting on them. Under the conditions of this experiment, therefore, it was found that in three weeks *P. infestans* had spread over 10 to 80 per cent of the surface area of the tuber, that in 11 cases it had spread into the eyes and traveled out into the sprouts, and that in the majority of the cases it spread most rapidly in the vicinity of the eyes.

This experiment was repeated under the same conditions, except that the sand was kept very dry and the tubers were held in it for six weeks. Without stating any of the details, which were much like those already given, it may be said that the fungus spread very slowly; and, while there was growth in some cases, in many the infected area remained as it was in the beginning. The tubers remained free from soft rots and germinated freely. From the results of this experiment it is very strikingly evident that to produce rapid spread of the mycelium in the tubers the sand must be kept well moistened.

In still another experiment the temperature was reduced instead of the moisture, the former being held at 4° to 6° C. In this case the fungus made little or no growth or spread in the tubers, and the potato gave as little evidence of activity, showing that both moisture and temperature exert a marked influence on the growth of the mycelium.

SPREAD OF THE MYCELIUM INTO THE SPROUTS

When it had been shown that the mycelium was alive in the tuber, at least at some point, its spread into the sprouts was studied. Three boxes (18 by 18 by 6 inches) were filled half full of soil which had never grown a crop of potatoes and which had been steamed for 40 minutes in an autoclave at 15 pounds' pressure. Twelve tubers were partially buried in each box, four of which were sound, the remaining eight being infected with *P. infestans* when harvested. The soil was well moistened with distilled water, and each box covered with a pane of glass. Each box in the series was held at a different temperature—that is, 15° to 20°; 20° to 22°; and 23° to 27° C.

The 8 infected tubers subjected to a temperature of 15° to 20° produced many sprouts, 5 of which became infected during the period under observation. The tubers subjected to 20° to 22° also produced 5 infected sprouts, these appearing during the first 14 days after planting. The greatest number of infections were obtained from the 8 diseased tubers held at 23° to 27°, 13 sprouts becoming infected during the first 14 days after planting. The checks remained free from infection. *P. infestans* seldom sporulated on the parent tuber unless the corky layer was broken, but it was very common on the basal portion of the sprouts growing from infected tubers. In many cases the eyes producing infected sprouts were cut out to learn whether the fungus was present in the tissues immediately surrounding them, and in every case it was found. This showed that the sprout infection was due to the spread of the mycelium and not to spores present in the air, for had the infection been due to spores the checks would have shown as high a percentage of infection as the diseased tubers. Infection by *P. infestans* occurred on sprouts of all sizes, from those barely visible to those nearly 1 inch in length. It was a very common occurrence to find the fungus sporulating first on the lower third of the sprouts, while on the upper two-thirds it was not apparent, but it required only one or two days for the remaining portion to become covered also, which indicates the rate of spread of the mycelium in the sprout tissue.

Naturally discoloration and decay followed the fructification of the fungus. Plate IV, figure 2, shows a potato with diseased and healthy sprouts. This is a late stage of sprout infection, and the tissues of the two infected shoots have blackened. The healthy sprout stands on a portion of the tuber showing no external evidence of the disease, while that part surrounding the diseased sprouts is infected with *P. infestans*. The fungus sporulated only on the sprouts of the diseased tubers, while those arising from the healthy tubers in the same box remained sound throughout, which makes it certain that infection was not by spores present in the air or soil, but by the migration of the mycelium in the tissues of the parent tuber.

This experiment was repeated and has been reported in full in an earlier paper (21). Except in one particular, the results were, in general, alike. In this case a sprout grew out near the surface of the soil from one of the infected tubers. This sprout became infected and the mycelium of *P. infestans* grew out from it into the soil for a distance of about 1 cm. This is not a usual occurrence and happens only when conditions are very favorable for the growth of the fungus. A slight decrease in the moisture content of the soil and the fungus is no longer in evidence, nor does it return if the original moisture condition is restored.

This experiment was again repeated on January 29, but only two sets of temperatures, 15° to 20° and 23° to 27° C., were used. The other set of temperatures was omitted because the supply of tubers was rapidly becoming exhausted, and, besides, it had been shown that temperatures between 15° and 27° were the most favorable. The results were, in general, like those already recorded and need no further consideration. From this series of three experiments, in which infected tubers were partially buried in moist, sterile soil, it is clearly shown that the mycelium of *P. infestans* in infected tubers spreads from the parent tuber into the sprouts, where it may sporulate freely.

Naturally the next step was to learn something as to the behavior of the infected tubers when wholly buried in the soil. To this end 12 sound tubers of the Irish Cobbler variety were artificially infected with a zoospore suspension held in contact with a sprout about one-fourth of an inch long by means of a ring of paraffin, as shown in Plate IV, figure 2. These tubers, together with 6 sound ones as controls, were buried 2 inches deep in a box of wet sterilized soil and placed in a saturated atmosphere at 23° to 27° C. The tubers had gone through the rest period, and in some cases the sprouts were 1 inch long. Eleven days after planting, 4 of the tubers had thrown up shoots. The remaining 8 were dug up to learn their condition, and it was found that in every case the fungus had spread into sprouts other than the one originally infected. Plate IV, figure 2, shows a tuber with the paraffin about the infected eye and the cluster of 5 sprouts at the bud end of the potato. One of the cluster, it should be noted, is free from infection. After the tuber was photographed it was cut and the discoloration typical of *P. infestans* was found at the base of the sprouts. That it was *P. infestans* was further shown by the production of spores and conidiophores on the discolored tissue. The fungus had spread from the initial point of infection over to the point where the cluster of infected sprouts originated from the parent tuber. The four shoots that came through the ground were allowed to remain until April 30, when they were dug up. These were found to be sound, while the parent tubers were totally decayed. The controls remained free from infection by *P. infestans* throughout and developed into normal plants.

In this experiment 4 of the tubers produced healthy plants, while the 8 others were completely overrun before any of the sprouts could reach the surface of the soil. This explains why seed potatoes infected with *P. infestans* give a poor stand. It also shows that the relation of the fungus to the sprouts is the same whether the tubers are wholly or only partially buried. Another significant fact brought out in this experiment was the presence of spores on the surface of the infected sprouts in the soil. This was especially true on sprouts attacked but not wholly killed.

GROWTH OF THE MYCELIUM UP INTO THE SHOOTS

When it became evident that the fungus could grow out into the sprouts from an infected tuber partially or wholly buried in the soil, experiments were outlined to ascertain whether it might not also grow up into the shoots. Thirty tubers were artificially inoculated by introducing living mycelium from pure cultures of *P. infestans* into a wound in each, and all were immediately planted in pots in the greenhouse, the same number of healthy tubers being planted as checks on the same date. None of the plants growing from these tubers showed any signs of infection with *P. infestans*, although they were watched carefully for 71 days, after which the experiment was terminated.

In another experiment 12 naturally infected tubers were planted in pots of steam-sterilized soil. The same number of healthy tubers were planted at the same time as checks. Only 4 of the 12 infected tubers came up, and 3 of these were much less vigorous than the controls. The spindly, sickly looking shoots that grew from the diseased tubers were watched for 47 days, but no sign of *P. infestans* was noted. The tubers were then dug up and found to be wholly decayed, but the stems were sound.

In a later experiment 200 naturally infected tubers were divided into four equal lots and planted directly on the greenhouse bench 1, 2, 3, and 4 inches deep, instead of in pots. An equal number of sound tubers were planted in a like manner as checks. Conditions were made highly favorable for the growth and development of the plants. Seven days after the tubers were planted, a few shoots were noted coming through the ground. The following germination was obtained (Table I).

TABLE I.—*Percentage of germination of potato tubers infected with Phytophthora infestans*

Number of days after planting.	Percentage of germination of seed planted—				Percentage of germination of checks.
	1 inch deep.	2 inches deep.	3 inches deep.	4 inches deep.	
26	33	39	33	13	96
38	39	39	45	44	98

Of the 78 plants that came up 21 were markedly abnormal, while the remaining 57 were quite sound. The sickly plants were covered with bell jars for several days at a time so as to make the moisture conditions more favorable for *P. infestans*, but not a single case of infection either on the basal portions of the stems or on the foliage was found, although the plants were examined daily until the vines died down.

From these experiments and others of a similar nature not mentioned here, it is plain that environmental conditions and the stage of development of the tuber planted determine whether the mycelium may or may not grow up into the shoots. The conditions prevailing in the ordinary greenhouse are not suited to the spread of the mycelium up into the stems.

Believing temperature and moisture to be the chief environmental conditions bearing on the development of *P. infestans*, experiments were made to determine the influence of these factors on the disease.

TEMPERATURE.—The influence of temperature was considered first. Three experiments were made, and, as all were practically the same, a description of one will suffice.

Five 12-inch pots were nearly filled with soil and steam-sterilized. On January 29, 1912, three tubers infected with *P. infestans* were planted 2 inches deep in each of three of these pots, and in the two remaining pots sound potatoes were planted as controls. Two of the pots were placed in a greenhouse where the temperature varied from 15° to 20° C., depending upon the time of the day; the third was placed in another greenhouse where the temperature ranged from 23° to 27° C. With each was placed a pot containing healthy tubers.

The first shoot to appear in the pots kept at 15° to 20° C. came up on February 6, or 8 days after the tubers were planted. The healthy tubers used as controls did not come up as soon as the diseased ones. They were more dormant at the time of planting. It has been observed by several investigators that tubers infected with *P. infestans* germinated sooner than healthy ones. In 12 days all of the diseased tubers had shoots up so high that the panes of glass covering the pots had to be removed. In order to keep the young potato plants in a moist atmosphere, a large bell jar was placed over each of the three pots. Careful examination was made daily. On March 18, or 45 days after planting, the plants were 7 inches tall, but showed no signs of *P. infestans*. At this time the plants held at 15° to 20° C. were dug up to learn the condition of the diseased tubers planted. Three were wholly decayed, while the other three were only half rotten and showed on the remaining portion the typical shrunken areas so characteristic of this fungus. All of the tubers in the control pot were sound. The three tubers partially decayed were now placed in a moist chamber in order to ascertain whether the fungus was still alive in them after being buried 45 days

and after having nourished several plants to partial maturity. Two days later an examination showed that spores and conidiophores were developing on two of the tubers; but no indication of infection was observed on either the leaves or stems which were placed in a moist chamber. Examination on the following day showed no further developments, and, as the potato plants were becoming very much discolored, the observations were discontinued. It should be noted at this point that the fungus was alive and able to sporulate on the diseased tubers after being in the soil for 45 days at a temperature between 15° and 20° C. Had the fungus been latent in the potato leaves and stems, as claimed by Massee (20), it should have developed. The most interesting and important fact brought out in this experiment was the production of healthy vines by a tuber having in it the mycelium of *P. infestans* which remained alive for 45 days.

The two pots which were kept at 23° to 27° C., one containing three infected tubers and the other three healthy tubers, came up a little earlier than those kept at 15° to 20° C. The first shoot came up on February 4, or 6 days after planting, and in 10 days all three of the diseased tubers had shoots up, some of them longer than others. The development of the tubers used as controls was several days behind that of the diseased tubers. Ten days after planting, the shoots were so tall in the pot containing diseased tubers that the pane of glass had to be replaced by a bell jar. The control was treated similarly. Nothing of special interest occurred until March 8, or 39 days after the tubers had been planted, when it was noticed that one of the small shoots growing from one of the diseased tubers appeared water-logged at and a short distance above the surface of the soil. It did not have the normal appearance common to the stems of the other seven shoots in the pot. Upon examination of the water-logged area with a hand lens, a white glistening growth could be seen on the surface. Some of this material was carefully removed and examined microscopically and proved to be spores and conidiophores of *P. infestans*. This infected plant was about 2 inches tall, spindly, light green, and less robust in appearance than some of the other plants in the same pot (Pl. V). The soil was carefully dug away from the stem, and a portion of it below the soil was found to be diseased. This portion gradually became darker as it approached the mother tuber, being brown and doubtless dead at the point of attachment. The parent tuber was nearly all decayed, except one small portion, which was still firm and from which the diseased shoot in question had developed. Free-hand sections made of the portion of the parent tuber where the stem was attached showed the presence of a nonseptate fungous mycelium which was undoubtedly that of *P. infestans*. The tissues of the stem nearest the mother tuber were softer than those higher up, which would indicate that the infection was of longer standing in that section of the stem.

The controls remained free from infection. Because of possible contamination, no further observations were made in the remaining plants in this pot.

This experiment was repeated, beginning February 22, 1912, but instead of large pots six boxes 18 by 18 by 6 inches were employed. Diseased tubers were planted in four of these and sound tubers in the remaining two. Eight were planted in each box, the conditions being exactly the same as in the preceding experiment.

On March 3, or 11 days after planting, one shoot was found just breaking through the soil in one of the two boxes at 23° to 27° C. It seemed perfectly normal both in color and in size, but on examination the next day both the shoot and the surface of the soil immediately surrounding it were covered with a white glistening fungous growth resembling that of *P. infestans*. Upon examining this growth microscopically it was found to be the potato fungus, as suspected. The mycelium on the soil had grown out from the infected shoot and seemed to be confined to the surface of the soil. The soil about the shoot was removed and the underground portion of the stem exposed. It was found to be water-logged just below the surface of the soil and was gradually becoming brownish as the parent tuber was approached. An examination of the parent tuber showed it to be badly decayed at one end, but quite firm at the other. The tissue of the tuber was examined at the base of the young shoot and showed the characteristic blackening due to *P. infestans*. After 48 hours in a moist chamber the fungus fruited profusely. Plate IV, figure 3, shows a cross section of the tuber and the infected shoot.

MOISTURE.—As stated earlier, moisture influences in some way the behavior of the seed tuber and the fungous mycelium contained therein. It was thought worth while to hold infected tubers in comparatively dry rather than very moist soil, as was done in the preceding tests. To this end 24 infected tubers with several living eyes each were planted in steam-sterilized soil on January 13, 1914, in a house where the temperature varied from 15° to 20° C. After 30 days they were covered with a glass-house and kept well watered. Ten of the tubers rotted in the ground before producing any shoots. Thirteen days later a small, spindly shoot growing from one of the tubers showed discoloration just at the surface of the soil. This infection spread upward and the fungus fruited the following day. The remaining 13 were allowed to stand two weeks more, but none of them became infected. When dug up, it was found that all the mother tubers were rotten except two. In these *P. infestans* fruited when the tubers were cut open and laid in a moist chamber, showing plainly that the fungus may remain alive in the parent tubers for at least two months under the conditions of this experiment and also that the mycelium may spread up the stem, even though the infected tuber is not held continuously in wet soil.

In order to test still further the effect of moisture on the growth of the fungus up into shoots, 12 vigorously germinating tubers of the Green Mountain variety were planted in only slightly moist, steam-sterilized sand. These tubers grew rapidly, and in six days some of the sprouts began to break through the surface of the sand. Twelve days later 2 of the 12 tubers were dead. The remaining 10 were potted in steam-sterilized soil and placed in a glasshouse where the soil was well watered and the humidity high. Nine days later one shoot of one of the tubers was badly discolored near the surface of the soil. The discoloration spread up the stem, and after two days the infected area bore conidiophores of *P. infestans* in considerable abundance. When the tuber was dug up, the shoot was found to be diseased throughout its whole length below the surface of the soil. Six days later another tuber showed an infected shoot like the one just described. The remaining 8 mother tubers were dug up two weeks later and found to be entirely decayed. These results tend to show that continuous high moisture content of the soil is not necessary for the growth of the mycelium in the tuber up into the stems. According to the results obtained in these experiments, the soil may be kept comparatively dry until the plants are up. Furthermore, under these conditions the tubers do not rot as rapidly, and a larger number of shoots are produced by each.

INFECTED SEED POTATOES THE CAUSE OF AN EPIDEMIC OF PHYTOPHTHORA INFESTANS

The relation between seed potatoes infected with *P. infestans* and the development of epidemics of the disease under field conditions has received consideration both in Europe and in America, but no one has yet been able to trace and establish beyond doubt the existing relationship. Both De Bary (1, 4) and Jensen (14) claim to have done so, but they made only limited tests in the open in gardens, where conditions are not always comparable to those existing in the field. A large number of field trials having been made with only negative results, coupled with the fact that the mycelium grew up into the stems under laboratory conditions, led the writer to make field trials. For this purpose a section of the country was chosen where this disease occurs annually—namely, northern Maine. Such a section should afford the environmental conditions suitable for the development of all phases of the disease.

FIELD STUDIES IN 1913

The land selected for the experiment had not grown a crop of potatoes for at least five years and had been in hay for the preceding four years. The infected seed planted was selected in the spring from five bins (1,200 bushels each) of potatoes, Irish Cobbler and Green Mountain varieties, grown in the vicinity of Houlton, Me., and held in storage

through the winter. The tubers selected showed various stages of infection; but none were used that did not show at least one living eye (bud). On June 6 the tubers were planted in a 2-acre field of potatoes somewhat isolated from adjoining fields, 256 being planted whole in two rows 8 rods long. In a third row 162 hills were planted with cut infected seed. Alternating with these, three rows were planted with healthy seed, Green Mountain variety, as checks. The seed was planted between 1 and 2 inches deep and the row hilled up so as to cover the sets from 3 to 5 inches. Continuous records were taken of the soil temperature by means of a self-registering Richard soil thermograph. A record was also kept of the rainfall, especially as to the date and approximate amount.

As would naturally be expected, the infected whole tubers sent up shoots more rapidly than the cut seed. Six of the whole tubers had shoots through the ground two weeks after they were planted. On July 6, 30 days after planting, 63 per cent of the whole infected tubers had shoots up; so also did 49 per cent of the cut infected seed and 97 per cent of the tubers planted in the three control rows. After July 6 the percentage increased very little in any of the foregoing cases. On this same date six of the whole diseased tubers that had failed to send up shoots were dug up for examination. Four of these were dead and nearly decayed, while the remaining two had two and five shoots, respectively, which were just ready to break through the surface of the soil. Plate VI, figure 2, shows the condition of one of these shoots immediately after digging. They were taken to the laboratory later and examined for spores of *P. infestans*, but none were found. Subsequently they were placed in a moist chamber overnight, and the next morning small patches of conidiophores bearing spores, which on microscopic examination proved to be those of *P. infestans*, were found scattered over the diseased areas. The infected shoots were very much like those obtained in the laboratory experiments discussed earlier. It should be noted that a few days before the plants were dug up a light shower of rain had fallen, which, it is believed, materially aided the progress of the fungus. These developments in the field experiments are wholly comparable with those in the laboratory, in which the sprouts were attacked and overrun by the disease before reaching the surface of the soil.

On July 13 a very interesting case developed in the row planted with infected cut seed. When the infection was first noted, the discoloration had extended up the stem of the plant only half an inch above the surface of the soil. There was no evidence of spores of *P. infestans*. The weather was clear and the humidity unusually low, a condition not favorable for sporulation of *P. infestans*. The plant was carefully watched the following day, but no evidence of sporulation could be detected. The next

morning, however, the fungus, which, on microscopic examination proved to be *P. infestans*, had fruited, a 500 c. c. beaker having been inverted over the plant in the evening. For three successive mornings after this date evidence of a new crop of spores of this fungus on the little plantlet was found (Pl. VII, figs. 2 and 3). Later the plantlet fell over, owing to destruction of tissue by the fungus and soft-rot organisms which followed. The stem was found to be discolored all the way down to the parent tuber, a distance of 6 inches. The plant was allowed to remain in the field in order to ascertain whether it might infect the foliage of surrounding plants, but no infection developed and the plantlet soon died and dried up. Conditions were probably unfavorable in this case for the development of secondary infections, owing to a poor stand in the row where this infected plant happened to be. This condition makes it necessary for the spores to be carried a greater distance than might have been the case had a higher percentage of the seed planted in this row grown. The stand in the row in question and also the infected hill are shown in Plate VII, figure 3, This case is of special interest in showing that no further development of the fungus occurred, although it did grow up the stem from the diseased parent tuber to the surface of the soil and sporulate.

It was not until July 25 that another case of infection by *P. infestans* was discovered on any of the six rows under experimentation. This case developed in one of the hills growing from a whole diseased tuber. The hill was a vigorous one with 13 shoots, all normal except 3. The smallest of these 3 was 6 inches tall, while the others were fully twice this height. The plantlet was well shaded by the others and was detected only on careful examination of the hill (Pl. VII, fig. 1). When first found on July 25, fully an inch of the stem above the surface of the soil was discolored and a hand-lens examination showed that a fungous growth was present. Some of this growth, collected on a slide and examined microscopically, proved to be spores of *P. infestans*. The weather for five or six days previous to July 25 had been rainy, cool at night, and quite warm in the day time, conditions highly favorable for the rapid growth and spread of the fungus, as demonstrated in the laboratory studies.

The infection spread up the stem into the petioles of the lower leaves and produced spores in abundance. On the 29th, or four days after the infection was first noted, two leaflets in the hill showed infection, and discolored areas appeared on the stems of three of the adjoining shoots about 2 inches above the surface of the soil. The next morning five new leaflets in the hill showed early stages of infection. These infections occurred on leaves in the lower third of the hill, and each day the number of infections increased on the foliage. On July 31 one leaflet was found infected near the top of a plant in one of the adjoining

check rows, and as there was no other evidence of infection in this entire row it seemed quite certain that the spores had come from the hill previously mentioned. On August 5, six days after this stray infection was first noted, 14 others were found immediately below it on the leaflets in the same hill. It seemed quite apparent that the spores had fallen from the infection above and infected the leaves below. The disease continued to spread rapidly until August 10, when a period of hot, dry weather for 10 days checked its development temporarily. At the end of this dry spell, however, it resumed activity, and an epidemic of blight was well under way in this portion of the field. All the plants in the plot, except those on a few short rows of a foreign resistant variety, were killed by late-blight before frost. Four other cases, similar to the one just described, developed between July 25 and August 4. The symptoms in all cases were the same and need not be repeated. In each case the spores produced by the initially diseased shoots infected adjoining foliage and became centers for the spread of the disease.

The plants in the three alternating rows planted with healthy seed were watched for evidence of stem and foliage infection as carefully as those planted with infected seed, as was also the rest of the 2-acre field, but in no case did any infections develop that could not be traced to the centers in the rows planted with infected seed. Of course, after the epidemic was well under way, the source of any single infection was unknown. The significant point and the one on which information was desired was the origin of the very early stages in the development of an epidemic and not the late.

The results of the field tests of 1913 may be briefly summarized as follows: (1) Only 63 per cent of the whole infected tubers and 49 per cent of the cut infected seed grew; (2) the mycelium in infected seed tubers responded the same way in the field as it did in the laboratory experiments; (3) shoots were found that became infected before they reached the surface of the soil; (4) others infected were able to break through the soil and become centers of foliage infection. On these dwarfed infected shoots the fungus fruited and infected the foliage, first in the same hill and later in those adjoining. In this way these hills became the centers for the development of an epidemic.

FIELD STUDIES IN 1914

It is well known that too much reliance can not be placed on the results of 1-year trials under field conditions. This is especially true when dealing with a fungus like *P. infestans*, which is very much influenced by environmental conditions. In view of this fact, it seemed desirable to repeat the field trials of 1913. In 1914, a plot of ground was chosen at Caribou, about 60 miles north of Houlton, Me., where conditions are fully as favorable for the development of late-blight as at

the latter place. A plot of ground was selected that had been lying idle in 1913, but which before had grown several crops of potatoes in succession.

Tubers of the Green Mountain variety showing all stages of infection by *P. infestans* were selected on May 25 from potatoes grown and held in storage throughout the winter in potato cellars at Caribou. Most of them were badly infected, as was natural to expect at this late date. Many had only one living eye, while others, of course, had several. Both whole and cut seed were planted in the same way as already described in the field tests of 1913. In one row 170 whole tubers were planted and 363 in two rows adjoining. On each side of these three rows two rows were planted with sound seed as checks, also of the Green Mountain variety. The planting was made on June 2, when the soil was drier than usual. There was very little rain until July 20, when an inch fell, but, as a whole, the season was drier than that of 1913 and therefore was less favorable for the development of late-blight.

An examination made on July 15 showed that 47.6 per cent of the whole infected tubers, 37.4 per cent of the cut infected seed, and 92 per cent of the healthy seed in the four adjoining rows came up. The low percentage of germination of the infected seed was probably due to two factors, the large amount of infection of the seed with *P. infestans* and the dry weather following planting. The infected seed rotted in the ground in the same way as described in the studies made in 1913.

The first case of infection by this fungus was discovered on July 22, two days after a heavy rain had fallen. It was in a hill grown from a whole infected tuber having nine shoots from 12 to 18 inches tall. Five of the smaller shoots were found to be infected at and below the surface of the soil. The soil was carefully removed from about the hill, and two of the five were found to be discolored all the way from the mother tuber up to the surface of the soil. The three others seemed to have become infected at the surface of the soil, probably by spores borne on the two shoots most generally infected. The infection of neighboring stems in the same hill above the surface of the soil was also noted in the field studies of 1913.

Two days later another hill, also grown from whole seed, was found to be infected. This had 14 shoots, varying from 10 to 18 inches high. The smallest shoot was discolored in the same way as described in the previous case, and upon further investigation the infection was found to extend down to the parent tuber. The fungous infection was evident by the glistening white growth on the stem just above the surface of the soil. None of the older shoots in this hill were infected at this date.

On July 26 one of four shoots in a hill grown from cut seed was found to be infected. These four shoots ranged from 6 to 14 inches in height. Two of the smallest shoots in this hill were infected with *P. infestans*. The

hills in the four check rows were watched as carefully as those in the two rows planted with infected seed, but no infections with *P. infestans* were found.

The development of foliage infection from the three centers described was gradual and wholly comparable to that described in considerable detail in the studies of 1913. It should probably be said in this connection that the first foliage infection was found on July 27, five days after the first case was discovered. By August 14 leaves within a radius of 10 to 20 feet from each center or station were infected with *P. infestans*. A bad epidemic of late-blight was in full swing throughout the whole 2-acre field by September 10. It is plain that the three centers above described formed the starting points for this epidemic. Other centers of infection may have developed subsequently, but no attempt was made to follow the later developments because of the constant recurrence of new foliage infections resulting from the infections about the original centers. The results of the field studies of 1914 confirmed in every way the results obtained in 1913.

The fact that a tuber infected with late-blight may cause an epidemic of the disease raises the question as to the rôle of infected tubers left in the field at harvest time. The majority of these are killed by frost, but a few remain in the soil or get covered during the digging of the crop and may pass through the winter in a living condition. Observations showed plainly that many tubers survived the winter of 1913 in Aroostook County, Me. The fields planted to oats in 1914 that had been in potatoes the previous season were well sprinkled with volunteer potato plants. It is common knowledge among the growers of northern Maine that some seasons volunteer potato plants are very plentiful. Their presence or absence is determined largely by the season, especially by the time and amount of snowfall.

POSSIBILITY OF CONIDIA OF PHYTOPHTHORA INFESTANS BORNE ON THE SEED TUBER REACHING THE SURFACE AND CAUSING FOLIAGE INFECTION

In 1876 De Bary (4) called attention to the possibility of conidia on ıe seed tuber being able to reach the surface and cause foliage infection. lecke (12) and Clinton (8) are inclined to believe they function more ıtensively than the mycelium in the seed tuber. Little is known about ıe production of conidia on infected potato tissue in the soil or their ılation to renewing infection from one year to another. For this reason was thought advisable to learn something about the possibility of the ungus fruiting on cut seed in the soil and whether the spores functioned.

To this end 31 infected seed pieces were planted in the usual manner n June 22, 1913, at Houlton, Me. The soil was quite dry, and the soil emperature ranged from 10° to 14° C. Three days later they were dug

up for examination, but no spores of *P. infestans* were found. They were again planted and the next day a rain fell, wetting the ground down to the seed potatoes. On June 30, four days after the second planting, the seed pieces were dug up again. Microscopic examination showed that spores and conidiophores of *P. infestans* were present on 26 of the 31 pieces and the growth of the fungus in seven cases was readily visible to the unaided eye. The spores were found to germinate freely in water. These seed pieces were again planted on July 1 and left in the ground for a period of 14 days. At this time careful examination revealed a limited number of spores on 5 of the pieces, but these spores did not appear to be normal; and when placed in water only 3 or 4 germinated. A search was also made for mycelium of *P. infestans* in the soil adhering to the seed pieces, but none was found. The plants that grew from these infected seed pieces were examined daily from the time they came up until the vines were nearly mature, but no infection by *P. infestans* appeared on the foliage.

The above experiment was repeated, beginning on July 2. In this test 14 diseased seed pieces were planted just after a light rain. Four days later they were dug up and examined; on 7 of the tubers spores of *P. infestans* were found. There was no indication that the mycelium was growing saprophytically in the soil adhering to the cut surfaces of the diseased pieces. The pieces were immediately replanted and allowed to grow throughout the season. On July 25 the stem of one of the plants showed infection at the surface of the soil. When dug up, it was found that all of the stem below the surface was diseased and also the parent tuber at the point where the stem originated. This tends to show that the mycelium grew from the parent tuber up into the young shoot and that the infection was not caused by spores in the soil. This plantlet stood in an exposed place and soon died. Spores were produced, however, and a leaf on an adjoining plant became infected. This spread slowly in the leaflet and only a few spores were produced. Finally the leaflet died and dried up and no further infections occurred on any of the plants in the same or adjoining rows. In both these experiments conidia were produced on the seed tuber, but none of them functioned in causing any infections.

In the spring of 1914 further tests were made at Caribou, Me. On June 4, 183 potato seed pieces infected with *P. infestans* were planted in accordance with common practice. The next day it rained. On June 7, 26 of the 183 seed pieces were dug up and examined for conidiophores and spores of the fungus. These were found on 9 of the pieces and the growth was abundant enough to be easily seen with a hand lens. On July 10, 12 more seed pieces were dug up and examined, but no evidence of fructification of *P. infestans* was found. The weather had been clear and warm the five preceding days and the soil was much drier than on June 7. It

may have been that spores were present somewhere on the cut surfaces, but they were not sufficiently abundant to be found even after long and careful search.

On June 20, 20 more of the 183 seed pieces were dug up and examined, but again neither conidiophores nor spores of the fungus could be found. The cut surfaces of the seed pieces in every case had either corked over or started to decay.

No mycelium could be found growing free in the soil about the diseased tubers. No evidence was obtained showing that the fungus continues to sporulate on the seed tubers in the soil. Spores are produced abundantly on the cut surfaces of tubers recently planted in moist soil only, but these disappear in the course of a week or 10 days. In an earlier part of this paper it has been shown that spores may be borne in considerable abundance on sprouts killed before they reach the surface of the soil. Whether these spores ever function in infecting other potato tissue below the surface of the soil has not been shown definitely by any of the earlier workers or by any of the writer's experiments.

There is still another possible source of conidial infection that should be mentioned in this connection. A common practice in northern Maine and other potato-growing sections is to feed the culls to hogs or to dump them in some out-of-the-way place about the farm. In the culls there are usually a considerable number of tubers infected with late-blight. When the skin is ruptured on these, the fungus may fruit. Spores borne in this way may reach potato foliage and lead to infection. Then again, as observed by the writer in numerous cases, tubers infected with late-blight are often dumped in some wet or swampy place on the farm. In two such cases an infection of late-blight was found on the mass of growing plants as early as July 25 and 29. It was impossible to determine how and where the infection originally started, but it was clear that the disease had made a good beginning. It is, of course, needless to say that if such cases developed near a potato field, it might readily become infected.

Whatever may be the possible relation of the conidia to the renewal of epidemics of *P. infestans*, two points are perfectly clear: (1) That spores are borne in the soil on the freshly cut surfaces of infected seed and on sprouts when the soil is sufficiently moist and (2) that the spores probably do not remain viable more than two to three weeks in the soil.

RATE OF SPREAD OF THE MYCELIUM OF PHYTOPHTHORA INFESTANS IN THE POTATO STEM

The rate of spread of infection in the potato stem is of interest because of its direct bearing on the growth of the mycelium from the diseased tuber up through the stem. Healthy plants from 20 to 55 cm. high were exposed to infection with *P. infestans* by spraying a spore suspen-

sion over the plants; and when infections developed on the stems their upper and lower limits were marked with india ink. The infected plants were kept in the greenhouse under conditions favorable for the normal development of the host.

Records were made of infections occurring anywhere on the stem from within 6 cm. of the ground to within a few centimeters of the top. Eight infections within 10 cm. of the ground were kept under observation for four days. The total upward spread of infection in these during the four days was 30 cm., or an average of 3¾ cm., and the downward spread was 21 cm., or an average of 2½ cm. Five infections from 10 to 20 cm. above the soil were studied. Two of these were allowed to continue for 48 hours, and the remaining three for only 24 hours. After two days the combined spread up the stem in the five cases was 11 cm., and down, 6 cm., the average spread up and down in each case being 2⅙ and 1⅙ cm., respectively. Three stem infections were studied that were more than 20 cm. above the soil; two were between 20 and 30 cm. and one 45 cm. After four days the total spread of infection upward was 23 cm. and downward 11 cm. The average upward growth was 7⅔ cm. and the downward 3⅔ cm.

It should be noted that in every case the spread of infection was more rapid up than down the stem and that the fungus progresses more rapidly in young than in old tissues. It is thus evident that it may require only a short time for *P. infestans* to spread sufficiently in the potato stem to reach the surface of the soil, once it is in the basal portion of the shoot. It is likewise quite probable that the fungus grows down the stem from the surface of the soil.

HISTOLOGICAL STUDIES OF THE RELATION OF THE FUNGUS TO THE POTATO STEM

The question arises as to which the mycelium uses when it grows up the infected stem, the cortex, vascular system, or central cylinder. A section of an infected stem always shows that the cortex is discolored, while the rest of the tissues are quite normal. The natural inference from this macroscopic evidence is that the mycelium used the cortex most extensively.

In order to get more exact evidence on this point, infected shoots were killed in various fixatives and were later sectioned and stained. In every case where the cortex was discolored, the cells had collapsed and took the stain very heavily, as shown in figure 1. In such cases the mycelium was not readily seen, and in the majority of cases it was absent. It was sometimes found, however, in the cells between the outer cambium layer and the inner cortical cells, but more often at this stage it was seen growing among the pith cells, as shown in figure 2. Where the cells of the cortex were more normal, or from ½ to 1 cm. above the border line between

healthy and diseased tissue, the hyphæ could be readily seen ramifying between the cells, as shown in figure 3. The mycelium can usually be found higher up in the stem in the cortex than in the pith cells when the disease is growing up the stem from the infected parent tuber. When the cortex has been destroyed it may be found in the pith cells. So far the author has seldom found the mycelium in the vascular system or the wood cells. Histological studies indicate that the mycelium of *P. infestans* spreads up the stem most rapidly in the cortical region when conditions are favorable for its rapid growth.

DEVELOPMENT OF EPIDEMICS OF PHYTOPHTHORA INFESTANS

One argument used persistently against the theory of resting mycelium being the means of perpetuation of *P. infestans* is the sudden and almost simultaneous outbreak of the disease over wide areas. It has seemed more plausible to many to imagine that some form of resting spore functioned in spreading the disease rapidly each year, as is known to be the case in related species. Massee (20) has questioned the capacity of the conidia of *P. infestans* to start an epidemic. He believes that epidemics start from mycelium of the fungus latent in the tissues which becomes active with the advent of favorable weather conditions.

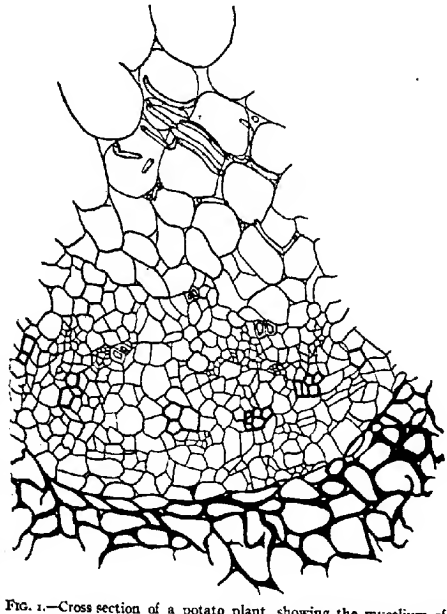

FIG. 1.—Cross section of a potato plant, showing the mycelium of *Phytophthora infestans*, which has killed the cells of the cortex and is a later stage than that shown in figure 3. The mycelium is present among the pith cells. The plant from which this cross section was made become infected like the one in figure 3.

In the fall of 1911 the following experiment was made at Madison, Wis., to learn something as to the development of an epidemic of *P. infestans* under field conditions, with special reference to the rôle played by conidia. It should be mentioned that this fungus seldom occurs in the vicinity of Madison, and, so far as known, it was absent from the State in 1911. The writer is sure it did not occur in the vicinity of Madison that year, and therefore his results were not complicated by its presence. On the even-

ing of August 17, 1911, after a spell of wet weather, two potato plants were sprayed with a suspension of spores of *P. infestans*, the spores having been taken from infected plants in the greenhouse. The inoculation of the two plants was made in the usual way and typical spots became visible in the course of five days. The amount of infection was not extensive. The ground was very moist, owing to the fact that several rains had fallen the previous week, and the weather was continuously cloudy from August 22, the date infection first appeared, until August 27.

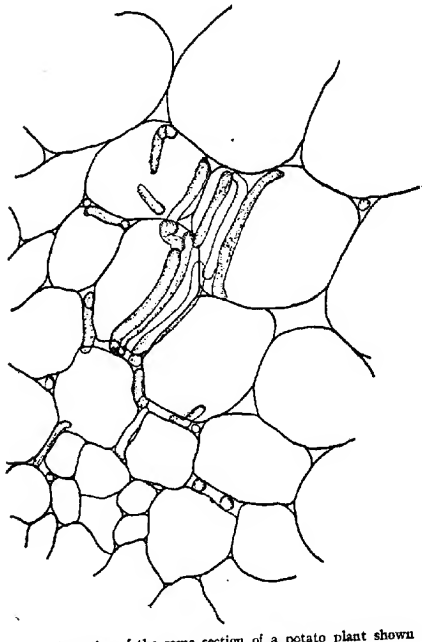

FIG. 2.—A portion of the same section of a potato plant shown in figure 1, showing the mycelium in the pith region of the stem.

On August 30 infections were found on two plants adjoining those artificially infected, and the next day four more plants immediately adjoining showed infection on several leaves. Careful examination showed no infection on any of the plants farther away. The new infections that had occurred on August 31 were on the six plants immediately surrounding the two artificially infected. The fungus had made no further spread in the half-acre potato plot. After August 30 new infections were daily found farther and farther from the two plants first infected, and on September 7 infected leaves could be found everywhere throughout the plot, though none of the vines were conspicuously blighted. By this time all the plants within a radius of 8 feet of the two plants initially infected were killed. Farther away the infection was much less in extent, though present in abundance. By September 12 the plot was very badly blighted; not a single plant anywhere was free from infection, and many were wholly dead. No further records except as to the time of harvesting and the amount of loss were kept when the tubers were harvested on October 10. Less than 50 per cent of the crop was fit to put in storage, and less than 10 per cent kept until spring, although held in good storage.

The conclusions to be drawn from this experiment are perfectly obvious. (1) An epidemic can be started by the infection of two plants in a field; (2) two infected plants can spread infection sufficiently to destroy the vines on a half-acre plot in 29 days. That a larger plot, indeed a field of many acres, could be destroyed by one infection is clearly evident.

It might be argued that these conditions were not typical of those occurring under field conditions. On October 14 a visit was made to the potato fields of western New York, where an epidemic was just starting in many of the fields. Infection centers like the one produced by artificial infection in the potato plot at Madison were in evidence in several fields. Another visit to the same fields early in November showed that they had been destroyed by an epidemic of late-blight.

The development of late-blight under field conditions was again followed in the fall of 1913 at Houlton, Me. Careful watch was kept on several fields in that vicinity. The first infection by *P. infestans* was found in the field on August 8, following a few days of wet weather. By going through nine different fields six other centers were found. One typical case will serve to illustrate the prevailing conditions at each center. The infected leaves were always the lower ones of the plant. At the center of the infected area the infections were much more numerous than elsewhere, probably about ten times as numerous. These centers of infection varied from 8 to 40 feet in diameter. If the centers had not become too large, a hill could usually be found that was nearly killed and which suggested strongly that it was the point where the primary infection originated. From August 15 to 28 the weather was hot and dry, and during this period the fungus made little headway. On the date last named a rain fell which facilitated the spread of the disease and caused it to become general though not markedly destructive in the

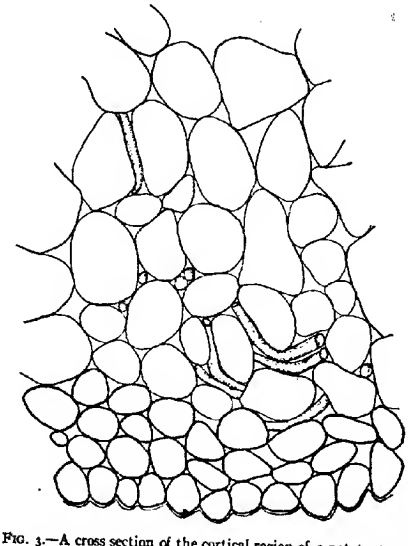

FIG. 3.—A cross section of the cortical region of a potato stem, showing the mycelium of *Phytophthora infestans*. This plant became infected by the mycelium spreading up the stem from the infected parent tuber. This is an early stage of infection, and the tissues of the cortex have not been killed.

fields not sprayed with Bordeaux mixture; in other words, epidemics were started from the small areas found early in the season. The spread of the disease was wholly comparable to the above-described developments on the small plot at Madison.

Last summer (1914) three similar infection centers were found in fields near Presque Isle, Me. Such a center is shown in Plate VIII, figure 2. The infected area is set off by a white line. The question naturally arises as to how these centers come into existence. Are they due to the planting of infected seed potatoes or to wind-blown spores? It is impossible to answer either of these queries positively, but in the light of evidence now at hand both are probable. There can be no doubt that seed potatoes infected with *P. infestans* are planted by the growers. This has been observed many times, and in one case 46 seed pieces infected with late-blight were taken from a single barrel of seed potatoes which were about to be planted. None of these were badly infected, but such specimens are more certain to produce infected progeny than those badly diseased, as the latter often rot in the ground.

It may well be, therefore, that these infected centers originate from the infected seed, even although the originally infected shoot is not found. This is probably due to its rapid death after the mycelium reaches the surface of the soil. It soon dries up and leaves little evidence of its presence behind. On the other hand, it is easy to understand how these infected centers might be caused by wind-blown conidia, but it is more difficult to explain their origin without making use of the progeny of infected seed tubers. Although it is not definitely shown how these infected centers originate, in the case of the experimental plots it was clear that they came into existence at the same time that the infected shoots developed. It is also known that seed potatoes infected with *P. infestans* are planted.

RELATION OF THE MYCELIUM IN THE SEED TUBER TO THE PROGENY

Logical as it seems that the shoots and plants produced by diseased tubers should become infected in the same way as the young sprouts, such has not been found to be the case by a large majority of the students of this problem. That the mycelium in the diseased tuber may renew infection from one year to another was first supported by experimental evidence in 1861 by De Bary (1). His evidence, however, was not generally accepted, and in 1876 Pringsheim (29) advanced the alternate-host theory. It should be recalled in this connection that De Bary announced the fundamental rediscovery of heterœcism in *Puccinia graminis* in 1865, which probably influenced Pringsheim (29) and many others in accepting the alternate-host theory as a possibility in *Phytophthora infestans*, where oospores were unknown and infected tubers failed to produce infected plants.

Pringsheim's theory, it must be conceded, won some consideration at the hands of practical growers. This is well illustrated in an early paper by Farlow (11) and an article by Jenkins in 1874 (13). The latter discusses 100 reports made by potato growers on the potato fungus. It is very apparent from these articles that clover or straw was thought by many to be an alternate host for *P. infestans*. This theory, as well as others equally fictitious, was not expounded after 1876, when De Bary published his second paper (4) on this subject. At this time he submitted further evidence supporting the perennial-mycelium theory.

De Bary's theory was not confirmed until about 26 years later, when Jensen (14) repeated De Bary's experiments and obtained infected plants which later became centers of secondary infection. He, like De Bary, worked only in the open, where accidental infection by conidia or by oospores is always possible and where such conditions as moisture and temperature are variable factors. In other words, the technique used by Jensen was no more refined than that used by De Bary 26 years earlier; and he, like De Bary (4), was unable sufficiently to define his method so that his results might be duplicated. In view of this fact it is not surprising that Jensen's researches failed to materially strengthen the perennial-mycelium theory.

During the last 25 years repeated efforts have been made by Boehm (6), Smorawski (32), Hecke (12), Clinton (8), Massee (20), Pethybridge (25), and Jones (17) to grow such diseased plants as were described by De Bary and Jensen from infected seed tubers, both under glass and in the open, but little confirmatory evidence has been obtained. This fact, coupled with the very important discovery by Jones (15), Clinton (9), and Pethybridge and Murphy (27) of resting spores borne by the late-blight fungus in pure cultures, has made the perennial-mycelium theory seem even more questionable. This feeling is liberally expressed by Clinton (8).

The fact that so many students have failed to show the relation of infected seed potatoes to epidemics of the disease may well be due to one or all of three factors: (1) Stage of activity of the tuber, (2) temperature, and (3) moisture of the air and soil.

It is well known that the tuber requires a rest period before it will begin to germinate. If an infected tuber is planted in moist, warm soil before this period has elapsed the tuber rots quickly, owing to the activity of *P. infestans* and soft-rot organisms. If, on the other hand, diseased tubers are held in cold, dry storage until late in the winter or early in the spring and then planted, the tuber makes considerable growth before it is overrun by *P. infestans* and soft-rot organisms. In several of our northern potato-growing sections potatoes are stored at temperatures ranging from 0° to 10° C. until only a short time before planting. The fact that *P. infestans* and soft-rot organisms make little or no growth at

this low temperature explains clearly how infected tubers are able to survive the winter season and are in a condition to make rapid growth when placed in the soil. The statements that tubers infected with *P. infestans* very largely rotted in the ground and that a large majority grew and produced normal plants are both very prevalent in the literature, and the author reports similar experiences in his own experiments. These discrepancies, however, may well have been due to the conditions under which the tubers were stored and their state of germination at planting time. Of course, as will be shown later, the influence of moisture and temperature after planting plays an important rôle.

From infected seed tubers growing rapidly the greatest number of infected sprouts and shoots were obtained in a saturated atmosphere at a relatively high temperature (23° to 27° C.). A temperature of 27° seemed even more favorable than 23° C. This is of interest in view of Vöchting's (35) results to the effect that the optimum for the growth of the potato tuber is about 27° and is not out of harmony with the optimum fixed by Jensen (14) for the growth of the mycelium in the potato tuber. How the fungus spreads in the stem and sprout tissues at temperatures between 23° and 27° C. has been described in an earlier part of this paper. The fungus not only traveled up the stem rapidly but also sporulated profusely at such temperatures. In a paper not yet published it is also shown that the growth of liberated zoospores is more rapid at 23° to 24° C. than at lower temperatures. This is true also where the vines have been inoculated with conidia and zoospores. Although no experiments have been made to establish the optimum for the growth of the mycelium in the diseased tuber, the data cited above show that the mycelium is very active at 23° to 27° C. Whatever may be the optimum for the mycelium in the tuber, this point is clear: That temperatures between 23° and 27° C. are more conducive to the growth of the mycelium than lower temperatures, other conditions being favorable.

Although the state of germination of the tuber and the temperature are important, they do not take precedence over moisture. It need hardly be mentioned that *P. infestans*, by virtue of its phylogeny, is a moisture-loving fungus. To the practical grower it is well known also that an epidemic of late-blight need not be feared in a dry season, while in our northern potato sections a wet season is a sure sign of such an epidemic. The mycelium grows very slowly and absolutely refuses to fruit in a dry atmosphere. It has been shown that the spread of the mycelium is materially retarded when tubers infected with *P. infestans* are buried in dry soil. Again, the necessity of moisture is well illustrated in the case of the isolated plantlet referred to. The fungus made little progress in the stem even after reaching the surface of the soil, and it was only by restoring a moist atmosphere that the fungus fruited. It has also been shown that a greater number of the infected

tubers produced young plantlets when they were allowed to sprout in comparatively dry soil.

De Bary (4) describes a case which is interesting in this connection and serves to emphasize the importance of moisture conditions. A potato plant was found which had become infected by *P. infestans* in the parent tuber. Portions of the stem just above the surface of the soil were infected and discolored, but dry weather prevented the fungus from progressing farther in the tissues or sporulating. This was surely a case where moisture checked the fructification of the fungus. Two similar cases, which are even more striking as showing the close relation of moisture and development of the fungus, are described in this paper. In these the fungus grew up the stem to the surface of the soil and infected the foliage, but the hot, dry weather checked its further spread.

It is not necessary that the optimum conditions for the growth of the fungus should prevail continuously. This is clear from the author's experiments where the tubers were started in dry soil and later transferred to wet soil and the fungus grew up the stem. Too much emphasis can not be placed upon the importance of environmental factors and the state of germination of the tuber in the production of diseased plants from seed infected with *P. infestans*. A combination of these three conditions is not always prevalent in the open nor in the ordinary greenhouse, which may well account for the accumulation of negative data. In this connection may be cited one of several experiments where over 300 tubers were planted in a greenhouse, where the moisture and temperature could not be readily controlled, and not a single infected plant was obtained. Clinton (8), Pethybridge (24, 25), and many others have reported similar results from extensive field trials.

In closing this portion of the discussion it should be pointed out that not all infected tubers give rise to infected shoots and become centers of foliage infection. In fact, only a small proportion function in this way, according to the studies of the author; nor has any method been worked out whereby an infected tuber can be made to give rise to infected plants such as are shown in Plates VI and VII. Whether the progeny of a diseased tuber will or will not become infected is determined by the response of the fungus and host, coupled with environmental conditions. It is known beyond all possibility of doubt, however, that a certain proportion of the diseased tubers planted under field conditions may produce progeny which becomes infected by the mycelium growing up the stem. Once above the surface of the soil, the fungus may sporulate and cause foliage infection on the initial and adjoining hills. Infection spreads rapidly from such an infection center and is the forerunner of an epidemic. Hecke (12) has also noted this early stage in the development of an epidemic. It seems logical to assume that these infection centers start from planted infected seed potatoes.

This method of perpetuation readily explains how *P. infestans* has spread from its native home in South America to every corner of the globe. As pointed out by Jensen (14), it was probably brought to Europe in the mycelial stage in seed potatoes. Likewise, it may well have gone to Australia, New Zealand, North America, and other parts of the world.

MYCELIUM OF PHYTOPHTHORA INFESTANS IN THE SOIL

That the mycelium might live over winter in the soil was possibly first suggested by Kühn (18), who arrived at this assumption because he was unable to grow infected plants from diseased tubers, combined with the fact that the potato fungus occurred year after year. This theory received support later at the hands of Brefeld (7) in connection with his excellent cultural studies of the smuts. He devoted some attention to *P. infestans* also and was probably the first to grow this fungus saprophytically in semipure cultures. It was this significant achievement that led him to support Kühn's theory.

Darnell-Smith (10) has studied the possibility of *P. infestans* living over in the soil. A large number of experiments were made by mincing infected tubers in the soil and planting it to potatoes. He also smeared spores on the tubers when planted, but in no case did he get any infection of *P. infestans*. Some recent experiments by Stewart (33) also bear directly on Brefeld's theory (7, p. 26). He planted healthy tubers in soil mixed with blighted vines and tubers and made conditions highly favorable for the infection of the growing potato plants. No infection of *P. infestans* was obtained.

According to the writer's studies, under certain conditions of moisture and temperature the fungus may grow and sporulate on the surface of the soil to a very limited extent, as described in an earlier part of this paper, but no evidence was obtained showing that it remains alive in the soil for extended periods of time. Jones, Giddings, and Lutman (17) have also recorded the fact that the fungus may spread from infected tissue out over the surface of the soil to a limited extent. Our increased knowledge of culturing parasitic fungi on artificial media, and especially of *P. infestans*, does not permit such deductions at the present time as were made earlier by Brefeld (7).

MASSEE'S LATENT-MYCELIUM THEORY

The early literature on *P. infestans*, then known as the "potato murrain," is full of interesting theories as to its origin. The literature is in every case naturally tinted with spontaneous generation and lack of information as to the life history of the fungus. Fully as interesting is a theory more recently advanced by Massee (20). He maintains that the usual explanation for the sudden appearance of *P. infestans* over wide areas by the dissemination of conidia is inadequate and that the fungus is

latent in apparently healthy potato plants. It is, of course, obvious that Massee makes two radical departures from well-established principles: First, that the rapid dissemination of spores is not sufficient to cause an epidemic; and, second, that mycelium remains latent in the potato tissues.

The development of an epidemic by means of conidia under field conditions has been carefully followed and described in an earlier part of this paper, and the results fully confirm Ward (36) and others. That conidia or asexual spores are able to cause epidemics in the case of a great number of parasitic fungi is well known and needs no further argument. Had Massee demonstrated histologically the presence of latent mycelium in the apparently healthy potato plant as a whole, the latent-mycelium theory would have been worthy of more careful consideration.

WILSON'S SCLEROTIA-LIKE BODIES OF THE POTATO FUNGUS

Another singular theory to account for the perpetuation of *P. infestans* is that proposed by Wilson (37). He believed he had found sclerotia-like bodies on the potato tuber and plant as a whole which were the resting organs of the potato fungus. This theory was later indorsed, strangely enough, by Plowright (28) and W. G. Smith (31). The latter stated that it was his conviction that the bodies Wilson found were of fungous origin, and possibly those figured by Martius (19). These sclerotial bodies were later proved by Murray and Flight (22) to be calcium-oxalate crystals.

Later Wilson (38) reported a more fictitious discovery, that of a mucoplasm existing in the potato plant, which was able to give origin to late-blight.

CONIDIA BORNE IN THE SOIL RENEWING INFECTION

De Bary early suggested that the fungus might perpetuate itself by means of the conidia, although he considered it very improbable that primary infection often, if ever, takes place in this way. Jensen (14) claims to have found a case where the shoots were killed before they reached the surface of the soil, and the spores on these shoots infected the stem of a healthy plant growing in close proximity. Clinton (8) also cites a case where conidia borne under wet cotton possibly functioned in causing infection in one of his pot cultures. In this paper are recorded further experiments showing that the fungus fruits with great ease on the cut surfaces of the seed tuber and on infected sprouts in the soil, although so far no case has been found where such spores functioned in producing infection above the surface of the soil. It is not impossible, however, that it might happen, and Hecke (12) records such a case.

As stated above, it is not improbable that spores produced on the cut surface of diseased tubers or sprouts may cause infection in some cases, yet the author can not hold with Hecke (12) and Clinton (8) that primary infection due to conidia occurs uniformly throughout a field. In an

earlier part of this paper it is shown how an epidemic developed by artificially inoculating two plants in a plot of potatoes in a section of the country where *P. infestans* did not develop that year and how plants immediately surrounding the two initially infected ones succumbed before any of the others at a greater distance, thereby giving rise to infection centers in the plot in which the vines were killed long before the rest and which increased until it included the whole plot.

Other cases are cited where similar centers known to have originated from the spread of the mycelium up the stem were found and carefully watched under field conditions during the growing seasons of 1913 and 1914. Furthermore, the development of *P. infestans* has been followed for the last three seasons, but no evidence has been obtained to show that it originates uniformly on the lower leaves throughout a whole field. In many cases, when observations are made early enough, the disease is found to originate at some one point and spread outward and radially.

RESTING SPORES OF PHYTOPHTHORA INFESTANS

Resting spores, or oospores, are produced by most of the species of Peronosporaceae. Their function, as is well known, is to bridge the fungus over periods unfavorable for its growth and development. Whether *P. infestans* has oospores has been a bone of contention for the last 60 years. Until recently, however, the prevailing opinion has been that oospores were not produced by this fungus.

During the last decade bodies resembling oospores have been found in pure cultures by Jones (15), Clinton (9), and Pethybridge (26). This discovery has doubtless influenced Pethybridge (25, p. 343) in making the following statement:

> It appears to be practically certain that the primary attack of blight each season is due to *spores*, but where these spores come from is not known with certainty, and whether they are similar to those produced on the potato foliage in warm, moist weather in the summer after the primary infection of the crop has taken place, or are of the nature of the thick-walled resting spores produced by species of Phytophthora allied to *Phytophthora infestans*, can not definitely be stated at present.

This statement plainly discredits the perennial-mycelium theory and suggests that spores, either conidia or oospores, function in renewing infection. That the mycelium in diseased seed tubers may renew an epidemic of late-blight has been clearly shown in an earlier part of this paper and needs no further argument.

Pethybridge (25) unfortunately does not define the spore that serves to perpetuate *P. infestans*. If he means conidia, there is little evidence to support his contention, as has already been pointed out. On the other hand, it must be conceded that the discovery of bodies resembling oospores in pure cultures of *P. infestans* must be seriously considered when discussing the overwintering of the fungus. At present, unfortunately, there is little positive evidence to support the oospore theory.

It is to be hoped that the recent researches on this problem will afford an angle of approach that will yield positive evidence in the near future.

In closing it should be pointed out that, although *P. infestans* rarely produces oospores in the potato plant, this should not be looked upon as abnormal. As shown in this paper, the production of resting organs is not necessary for the hibernation of the fungus. The mycelium is quite sufficient. There are many species closely related to *P. infestans* that produce few resting spores on certain of their hosts. These may perpetuate themselves from one season to another by means of the living mycelium in the perennial parts of the host plant in much the same way as already described for *P. infestans*. The sparing production of oospores and the hibernation of the mycelium are therefore not uncommon in several species of this family.

SUMMARY

It is clear from the author's experiments that the mycelium of *Phytophthora infestans* spreads in the tissues of the potato tuber and finally reaches the sprouts. The growth of the fungus is retarded when diseased tubers are held in dry soil or at temperatures below 5° C. Infected tubers rot rapidly when placed in warm wet soil. This explains the wide variation in stand obtained by earlier writers. A temperature of 23° to 27° C. and a well-watered soil were found to be the most favorable for the mycelium to spread in the tuber and grow out into the sprouts, both when partially and when wholly covered with soil. Under these conditions the sprouts may become infected from 4 to 20 days after planting, regardless of their size and age. The time required is doubtless influenced by the proximity of the mycelium to the buds and the external conditions.

The mycelium of *P. infestans* may remain alive in seed tubers planted in the soil for at least 45 days, and it is very possible that under conditions less favorable for the soft rots which follow *P. infestans* in the tuber the fungus may live much longer. None of the author's results or observations tend to show that the potato fungus is *latent* in the stems and leaves of plants growing from diseased tubers, as stated by Massee (20).

Laboratory tests showed that the fungus infects not only the sprouts but also the shoots that break through the soil. The mycelium grows from the tuber into the stem, where it travels up to the surface of the soil and sporulates, as held by De Bary (4) and Jensen (14). This usually takes place in the small dwarfed shoots in a hill.

Potato tubers infected with *P. infestans* used for seed purposes and planted under field conditions may cause the development of an epidemic. The mycelium grows from the parent tuber up into the stem exactly as shown in the laboratory experiments. It later sporulates and foliage infection results. Ten such cases were found and followed

in northern Maine during the growing seasons of 1913 and 1914. All except two of these became centers for foliage infection, and severe epidemics of *P. infestans* followed.

Conidia of *P. infestans* may be borne on the cut surfaces and sprouts of tubers when planted under field conditions. As the cut surface corks over or the tuber decays, the fructification of the fungus decreases. Spores taken from tubers two to three weeks after they were first planted showed only limited germinating capacity. No evidence was obtained tending to show that the conidia borne in the soil are instrumental in starting foliage infection.

The mycelium of *P. infestans* spreads most rapidly in the cortical tissues of the stem, where it travels up more rapidly than down.

Epidemics of late-blight may start from a single shoot or hill naturally or artificially infected with *P. infestans*. The infection spreads radially from the initial point of infection during the early stages of the development of an epidemic. These spots of infection in the fields probably come into existence through the planting of seed potatoes infected with *P. infestans*.

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PLATE IV

Phytophthora infestans: Infection of potato tubers

Fig. 1.—Cross section of a tuber which was infected with *P. infestans* and was planted in the greenhouse in rather dry soil. After two months it was dug up and found to be firm and containing living mycelium of the fungus.

Fig. 2.—This tuber was inoculated at the eye surrounded by the paraffin ring. The mycelium ran through the tissues and grew out into four of the sprouts at the bud end of the tuber.

Fig. 3.—Cross section of an infected tuber planted in sterilized soil in the greenhouse which developed a shoot that became infected through the parent tuber.

Fig. 4.—The small stunted shoot which grew from this infected tuber shows the progressive discoloration caused by *P. infestans* growing up the stem.


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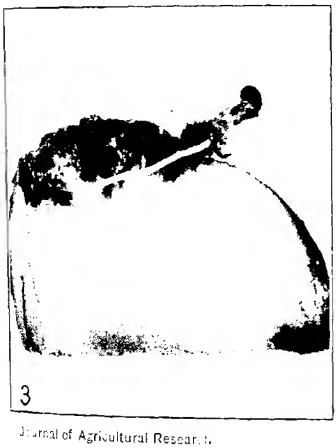
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PLATE V

Potato plant showing infection by *Phytophthora infestans*

Three diseased tubers were planted in the greenhouse and held at 23° to 27° C. for 36 days. At this time the small plant in the foreground became infected with *P. infestans*.

PLATE VI

Phytophthora infestans: Infection of potato shoots and plantlets

Fig. 1.—This shoot grew from a diseased tuber planted in the greenhouse under field conditions. Note the discoloration typical of *P. infestans* running up the stem.

Fig. 2.—This shoot, which had not reached the surface of the soil, grew from an infected tuber in the field.

Fig. 3.—This plantlet was the progeny of a diseased tuber planted in the open. It should be compared with the shoot shown in Plate VI, fig. 1, produced in the greenhouse. The same symptoms developed in the field as obtained in the laboratory.


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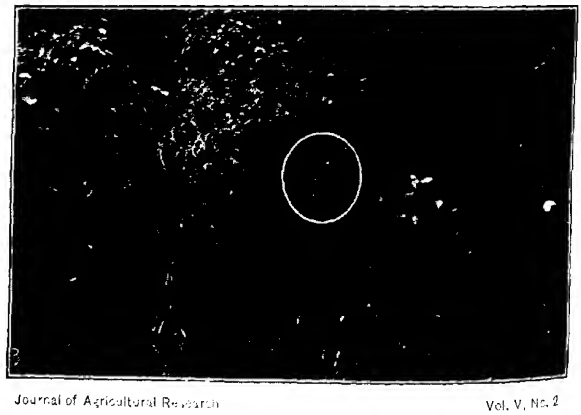

PLATE VII

Phytophthora infestans: Infection of potato plants

Fig. 1.—A hill of potatoes having 13 shoots grown from a whole infected tuber in the field. The smallest shoot, indicated by the arrow, became infected by the mycelium growing up through the stem from the parent tuber.

Fig. 2.—In this hill with two shoots the fungus has reached the surface and killed its host.

Fig. 3.—This shows the hill illustrated in Plate VII, fig. 2, in its position in the row where it grew. Notice the poor stand obtained by planting infected seed potatoes. This hill did not become a center for the spread of *P. infestans*, owing to its isolation in the row and early occurrence.

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PLATE VIII

Phytophthora infestans: Infection of potato plots

Fig. 1.—A corner of the plots where infected seed potatoes were planted. An epidemic originated from shoots which became infected through the parent tuber. The four rows of potatoes that still remain standing were of a resistant variety.

Fig. 2.—The area within the white lines shows a spot where infection is much more prevalent than in the surrounding plants. This spot functioned as a center for the development of an epidemic of late-blight in this field.


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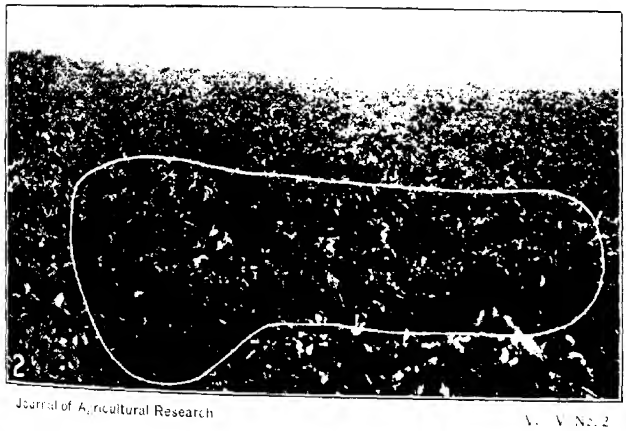
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